

## INVITED COMMENTARY

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The Swedish Twin Registry, which is administered by the Department of Medical Epidemiology and Biostatistics at Karolinska Institute in Stockholm, Sweden, is the largest in the world, containing approximately 86,000 twin pairs. This robust registry was begun in the 1960s by two Swedish investigators, Lars Friberg and Rune Cederlöf, who utilized twin information dating back to 1886 to study risk factors for cancer and cardiovascular diseases while controlling for the genetic propensity to disease. The Registry links easily with the National Patient Registry, which has data on all public inpatient care in Sweden, to identify particular disease processes such as abdominal aortic aneurysms (AAA).

The current study, by Wahgren and colleagues, utilizes information gained from this population-based monozygotic and dizygotic twin data. This study is unique in its characterization of the genetic and environmental influences on the development of AAA. Dr Wahgren has provided the most complete and convincing epidemiologic evidence to date for the existence of heritable contributions to aneurysm formation. The major findings of this study are that concordances (identical genetic traits) and correlations (agreement in continuously varying traits) are higher in monozygotic twins than in dizygotic twins, indicating true genetic effects. Data from this study demonstrated that the identical twin of a monozygotic twin with an AAA has a 24% chance of having an AAA. Alternatively stated, the twin has a risk of AAA that is 71 times greater than if the first twin did not have an AAA. Interestingly, gender was not influential, most likely because of the small number of women in the study.

Twin births are relatively uncommon, as they represent only 3% of all births in the United States. However, much data can be

gained from the study of the presence or absence of complex traits and coexisting environmental influences in this small cohort. We know that environmental factors such as smoking, nutrition, dyslipidemia, infections, and random factors (toxic exposure, spontaneous mutations) may contribute to AAA formation. The twin model is unique in that several key assumptions are made including equivalent environmental exposure and removal of the influence of age. Of the 265 concordant twin pairs (both affected with AAA) in the Swedish Registry, genetic effects accounted for 70% and non-shared environmental effects accounted for the other 30% (no effect from similar or shared environmental effects) of the phenotypic variance. It is well known that first-degree relatives, particularly siblings of AAA patients, have an increased risk for AAA. By studying concordant and discordant twins, these investigators have removed doubts about age variability or differential environmental exposures occurring in childhood and early adulthood.

Still, the population in Sweden may be different than more heterogeneous populations that exist elsewhere in the world, limiting the value of this study. The government of Sweden provides equal access to medical care and basic education. Even so, inequalities exist where health status may be a function of socioeconomic standing. However, for Sweden and the United States, as well as other countries, this study provides convincing evidence for genetic contributions to AAA development and perhaps evidence for a stronger role in screening siblings of affected persons. Furthermore, Sweden's robust data repository remains an invaluable resource for the scientific study of disease processes in the twin population and is an example for others to follow.